The patient was treated with daunorubicin, cytarabine, and etoposide with disappearance of blast cells from the peripheral blood and the marrow. However, her menorrhagia recurred in August 1994 and an endometrial biopsy was done, showing a diagnosis of granulocytic sarcoma (Fig. 1). At that time, peripheral blood counts were: hemoglobin 10.7 g/dL, platelets 22×10^9/L, and leukocytes 4×10^9/L with rare blast cells. Bone marrow examination showed no excess of blast cells. In September 1994, she developed a right cervical mass which was also shown to be granulocytic sarcoma on fine-needle aspiration cytology. Rapid leukemic transformation ensued after two weeks, and she was treated with intensive chemotherapy and local irradiation to the right cervical region. This was followed by persistent cytopenia, and the patient died of septicemia in November 1994.

Granulocytic sarcoma is uncommon in patients with MDS, and can occur at presentation or during blastic transformation [2]. Granulocytic sarcoma can involve virtually any anatomic site in the body, such as the lymph node, skin, nasopharynx, and gastrointestinal tract [4]. Cutaneous involvement is commonest for granulocytic sarcoma occurring in the setting of MDS [2]. However, granulocytic sarcoma involving the uterus is distinctively rare [4,5], and like granulocytic sarcoma involving other sites, the lesion is often misinterpreted as malignant lymphoma on histologic examination [5]. Our patient has an unusual presentation, with granulocytic sarcoma developing in the uterus and at a time when no evidence of leukemic transformation was observed in either the peripheral blood or bone marrow. In female patients with MDS/AML and menorrhagia, the bleeding disorder is often attributed to platelet dysfunction and thrombocytopenia. In occasional cases, however, this may be due to granulocytic sarcoma in the uterus. Detailed gynecological examination is therefore warranted, particularly when the bleeding is not explainable by the degree of thrombocytopenia.

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Chronic Consumption Coagulopathy and Popliteal Aneurysm

To the Editor: Chronic intravascular coagulopathy is a well-known cause of thrombocytopenia and may exceptionally complicate thoracoabdominal aneurysms [1]. We report such a coagulopathy in a patient with a popliteal aneurysm.

A 94-year-old man was referred for moderate thrombocytopenia. The patient had no medical history and no bleeding tendency. Physical examination was noticeable for the absence of arterial pulse in the left foot and a palpable left popliteal mass. Blood examination showed a moderate thrombocytopenia (60–80,000/mm^3), hemoglobin was 150 g/l, and leukocytes were 5,700/mm^3 with a normal differential count. Fibrinogen was 3 g/l, activated partial thromboplastin and prothrombin times were normal, fibrin degradation products were positive (80–160 μg/ml, N < 20 μg/ml),

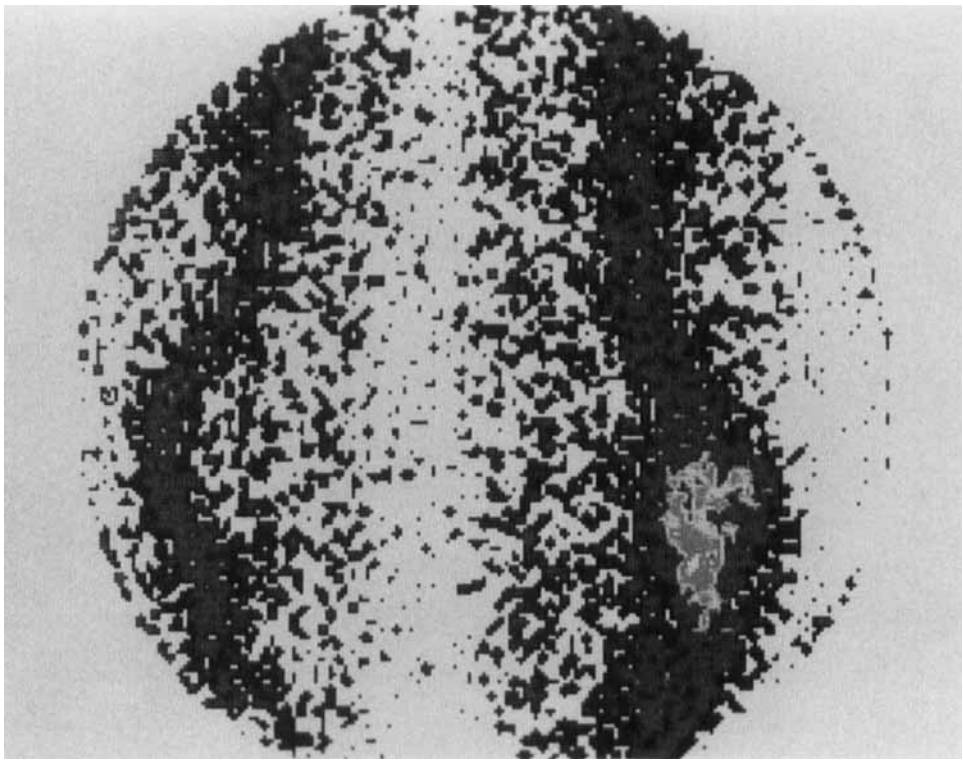

Fig. 1. Indium 111-labeled platelet scintigraphy.

liver and renal functions were normal. Arterial ultrasound and computed tomography scan of the legs confirmed the presence of a voluminous popliteal aneurysm (diameter: 50 mm, length: 100 mm), with partial mural thrombus and a residual lumen of 15 mm. Indium 111-labeled platelet scintigraphy showed increased accumulation of radioactivity over the aneurysm (Fig. 1). No surgery was performed because of the patient's advanced age.

Increased fibrin split products may be present in as much as 40% of patients with aortic aneurysm. A more severe consumption coagulopathy with thrombocytopenia, however, is observed in only 4% of cases [1]. Usually, these aneurysms are extensive, involving the thoraco-abdominal aorta [1–3], and the pathogenesis of the consumption coagulopathy appears to be a continuous dynamic process of intravascular coagulation with platelet consumption and secondary fibrinolysis [1,4]. In this patient, no aneurysm was detected on the thoracoabdominal aorta, and the indium-labeled platelet scintigraphy showed that the coagulopathy initiated in the popliteal aneurysm. To our knowledge, this is the first time such an observation has been made, but this popliteal aneurysm was unusually large and thrombosed. This observation suggests that size and shear flow conditions inside the lumen, rather than location of the aneurysm, might be critical for the development of chronic consumption coagulopathy.

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Persistent Polyclonal B Lymphocytosis

To the Editor: We report a case of persistent polyclonal B lymphocytosis which presents, in contrast with others communicated in the literature, certain differences.

A 35-year-old woman was admitted to hospital 2 years after presenting with a persistent lymphocytosis whose cause had not been investigated during this period. Prior medical history disclosed only psoriasis and duodenal ulcer. She was a heavy smoker (20 cigarettes/day) and had complained of asthenia during the preceding 4 months. Physical examination was negative. Peripheral blood showed: hemoglobin 130 g/l; white blood cell count 11×10^9/l (24% neutrophils, 70% lymphocytes; 3% monocytes; 3% eosinophils); platelets 203×10^9/l. Morphologic study of the lymphocytes showed 60% atypical and 5% binucleated forms (Fig. 1). No evidence for an active or persistent viral or rheumatologic disease was found (only evidence of a previous Epstein-Barr virus [EBV] infection was detected, viral capsid antibody IgG 1:640). Immunophenotyping showed that the lymphocytosis was of the polyclonal B-cell type: CD19+, CD20+, CD21+, CD22+, and both kappa and lambda light-chains were expressed, with a ratio of 6/11. Serum immunoglobulins revealed decreased levels of IgA and IgG and high levels of polyclonal IgM (7.37 g/l). Bone marrow aspirate showed slight lymphocytosis (30%), with 10% atypical forms. The patient was followed-up for 4 years without any change in the physical examination,

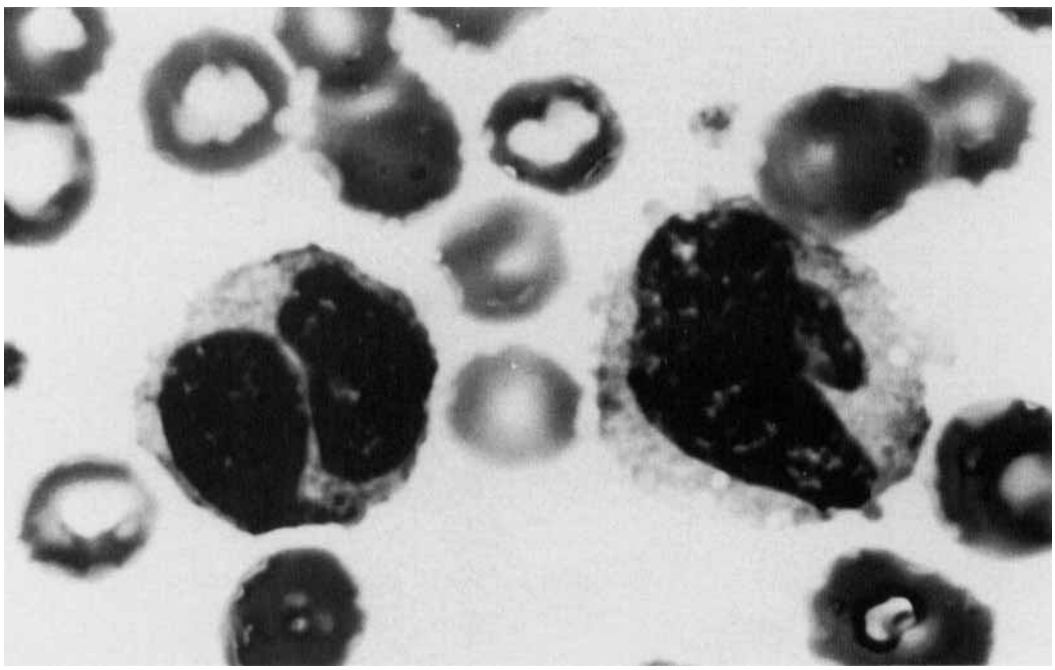

Fig. 1. Binucleated lymphocyte in peripheral blood.